Management of surgical patients receiving anticoagulation and antiplatelet agents

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Background: Temporary interruption of long-term anticoagulation and antiplatelet therapy during surgical procedures exposes patients to thrombotic risk. Continuation of these agents, however, is associated with an increased risk of bleeding. Managing anticoagulation can be a particular challenge in the emergency setting.

Methods: A literature review of published articles sourced using the keywords heparin, warfarin, perioperative, antiplatelet, aspirin and surgery was undertaken. A management plan for all likely situations was developed.

Results and conclusion: Based on an individual assessment of risk factors for arterial or venous thromboembolism and the risk of perioperative bleeding, it is possible to form an anticoagulant and antiplatelet management plan likely to achieve a low incidence of bleeding and thrombosis. A multidisciplinary approach is desirable.

Introduction

There is not yet a consensus on the appropriate perioperative management of patients receiving anticoagulants and/or antiplatelet agents who are about to have surgery. Various protocols aimed at minimizing the risk of thromboembolism and bleeding have, however, been proposed. The best strategy probably involves initial assessment and risk stratification based on patient- and procedure-related risk factors for thrombosis and bleeding.

Assessment and risk stratification

Thrombotic risk relating to the indication for anticoagulation

The main indications for anticoagulation are atrial fibrillation, venous thromboembolism and artificial heart valves. The thrombotic risk is defined as the risk of thromboembolism for each condition if not on anticoagulant therapy.

Atrial fibrillation

Atrial fibrillation is associated with an increased risk of stroke and thromboembolic complications, a risk that is substantially reduced with antithrombotic therapy. Patients with lone atrial fibrillation and no other risk factors have the lowest risk of stroke (less than 1 per cent per year)[1]. The incidence is greater in the presence of risk factors for stroke such as *c*ongestive heart failure, *h*ypertension, *a*ge 75 years or older, *d*iabetes mellitus and a history of *s*troke or transient ischaemic attack (CHADS). CHADS-2 scoring is described in *Table 1*[2]. The risk of thrombosis is further increased in the presence of mitral stenosis and prosthetic heart valves[3]. Most patients, however, fall into a subgroup with the above risk factors but without previous transient ischaemic attacks or strokes; such patients have a risk of stroke of 3–7 per cent per year[4].

Thromboembolism

Patients with a history of recurrent venous thrombosis are usually receiving long-term anticoagulation as they are at high risk of future thrombosis. This also applies to those with antiphospholipid syndrome with a history of clots. Patients with a deep vein thrombosis within the preceding month are also considered at high risk of recurrent thromboembolism; they have a recurrence risk of 40 per cent per year without anticoagulation[5]. During

Table 1 CHADS-2 score[2]

Assign one point each for
    Presence of congestive heart failure
    Hypertension
    Age 75 years or older
    Diabetes mellitus
Assign two points for history of stroke or transient ischaemic attack

CHADS, *c*ongestive heart failure, *h*ypertension, *a*ge 75 years or older, *d*iabetes mellitus and a history of *s*troke or transient ischaemic attack. The stroke rate per 100 patient-years without antithrombotic therapy increases by a factor of 1·5 for each one-point increase in CHADS-2 score.

the second and third months of treatment, the risk is about 10 per cent per year (intermediate risk), and after 3 months of oral anticoagulation the overall risk of recurrence is estimated at 1·5 per cent per year (low risk)[5].

Artificial heart valves

Patients with mechanical heart valves who are not anticoagulated have a 15 per cent mortality risk from embolic stroke and a 70 per cent risk of major neurological deficit[6–9]. The incidence of major thromboembolism in patients with mechanical heart valves is approximately 4 per cent per year, but this is reduced by almost 75 per cent with anticoagulant therapy[5,6]. This thrombotic risk is related to the valve site, the design of the valve and a previous history of thrombosis. Artificial valves in the mitral position produce a greater risk of thrombosis than those in the aortic position owing to increased vascular stasis around the mitral valve[3,7]. Older-generation valves such as caged-ball valves (for example Starr–Edwards) have a greater thrombotic risk than bileaflet valves (such as St Jude), whereas the single-leaflet tilting-disc type (for example Bjork–Shirley) has an intermediate risk[7,8]. The thrombotic risk is increased in those with more than one prosthetic valve[3,7]. A history of previous arterial or venous thrombosis also puts these individuals at greater risk of further thrombosis[3].

Thrombotic risk of the procedure

The risk of postoperative venous thromboembolism with major surgery may increase 100-fold in the absence of thromboprophylaxis[5]. There is even a smaller, but definite, risk associated with simple laparoscopic procedures[10]. The risk of arterial thromboembolism is also greater in those not receiving anticoagulation in the perioperative period[10,11]. Dunn and Turpie[12] carried out an analysis of 31 reports of the perioperative treatment of patients receiving oral anticoagulants, and found arterial thromboembolism and stroke rates of 1·6 and 0·4 per cent respectively[12].

Bleeding risks owing to patient characteristics

The history is the most important tool for assessing the risk of surgical bleeding[13]. A history of transfusion with previous invasive procedures (such as dental extraction, surgery), childbirth or trauma is helpful in identifying a congenital haemorrhagic disorder. Acquired bleeding problems secondary to liver and renal failure, and disseminated intravascular coagulation, can also put patients at risk of bleeding. Furthermore, the use of concomitant antiplatelet and non-steroidal anti-inflammatory medications increases the chance of perioperative bleeding.

Bleeding risk of the procedure

Bleeding risk varies widely depending on the type of surgical procedure, and each surgical subspecialty identifies the risk for each procedure accordingly. Neurosurgery, vascular surgery and procedures such as renal biopsy are not only potentially haemorrhagic but also carry a high morbidity if severe haemorrhage ensues. A reasonable estimate of the incremental risk of major bleeding with the use of perioperative low molecular weight heparin (LMWH) is 0–2 per cent for non-major surgery and 2–4 per cent for major surgery[14,15]. The case fatality rate of a major bleeding episode is 3–8 per cent[16,17]. Factors such as the location and extent of surgery, and the accessibility of means of controlling bleeding by packing and suturing, may also influence management[11].

Based on the thrombotic and bleeding risks, patients receiving anticoagulation may be categorized into different risk groups (*Tables 2* and *3*), and the anticoagulation may be planned for the preoperative and postoperative periods (*Fig. 1*). Throughout this article 'oral anticoagulation' can be taken to mean 'warfarin'.

Preoperative period

The planning of perioperative anticoagulation should ideally start in a preoperative assessment clinic. A chart with recommendations from the surgeon after discussion with a haematologist and anaesthetist if necessary can be useful (*Fig. 2*). It is important at this stage to discuss the risks and benefits of stopping or continuing anticoagulation with the patient, and also to obtain informed consent.

Based on the risk assessment, those who fall into the low bleeding risk group (for example dental extractions) may continue anticoagulation, especially if the international normalized ratio (INR) is within the therapeutic range[22]. If the INR is higher, it should be allowed to return to within range before the procedure.

Table 2 Examples of bleeding risk associated with various interventions

High risk
Neurosurgical operations
Complex ophthalmic operations
Complex cardiac operations
Intermediate risk
Abdominal operations
Genitourinary operations
Extensive oral surgery
Thoracotomy
Joint replacement
Low risk
Dental procedures
Dermatological procedures

Those in the intermediate bleeding risk groups (such as abdominal surgery) need further characterization based on their thrombotic risk. An associated low thrombotic risk means discontinuation of the oral anticoagulant before the procedure and administration of LMWH only if the surgical procedure is associated with significant thrombotic risk. Patients in the intermediate and high thrombotic risk groups require their oral anticoagulation to be stopped and substituted with LMWH. The latter should be administered in a prophylactic dose for the intermediate group and a treatment dose for the high-risk group.

Surgical procedures that may be associated with a high risk of morbidity and mortality because of bleeding (for example neurosurgery) require intravenous unfractionated heparin (UFH) instead of LMWH (as discussed below) and possibly an inferior vena cava filter if there is an additional high thrombotic risk.

Table 3 Thrombotic risk stratification[5–8,12,18–21]

High risk
Atrial fibrillation
With prosthetic heart valve in any position
With a history of cardiac embolism (TIA or stroke)*
With rheumatic mitral valve disease
Thromboembolism
Recurrent (two or more) arterial or idiopathic venous thromboembolic events
Thromboembolic event with a hereditary or acquired hypercoagulable disorder such as factor V Leiden or antiphospholipid antibody syndrome
Venous or arterial thromboembolism within the preceding 3 months
Acute intracardiac thrombus visualized by echocardiography
Prosthetic heart valve
Recently placed mechanical valve (less than 3 months)
Older-type valve (single-leaflet tilting disc or ball in cage) in mitral position
Aortic single leaflet
Two prosthetic valves simultaneously
Intermediate risk
Atrial fibrillation
Without a history of cardiac embolism but with risk factors for it*
Thromboembolism
Venous or arterial thromboembolism more than 3 months but less than 6 months previously
Multiple (two or more) strokes or transient ischaemic attacks without risk factors for cardiac embolism
Prosthetic heart valve
Newer-type valve (e.g. St Jude) in mitral position
Older mechanical valve model in aortic position
Aortic valve and more than two risk factors for cardiac embolism
Low risk
Atrial fibrillation
Without multiple risk factors for cardiac embolism*
Thromboembolism
Venous or arterial thromboembolism more than 6 months previously
Cerebrovascular disease (such as carotid atherosclerosis) without recurrent strokes or transient ischaemic attacks and risk factors for cardiac embolism
Prosthetic heart valve
Newer-type valve in aortic position

*See *c*ongestive heart failure, *h*ypertension, *a*ge 75 years or older, *d*iabetes mellitus and a history of *s*troke or transient ischaemic attack (CHADS) score in *Table 1*. TIA, transient ischaemic attack.
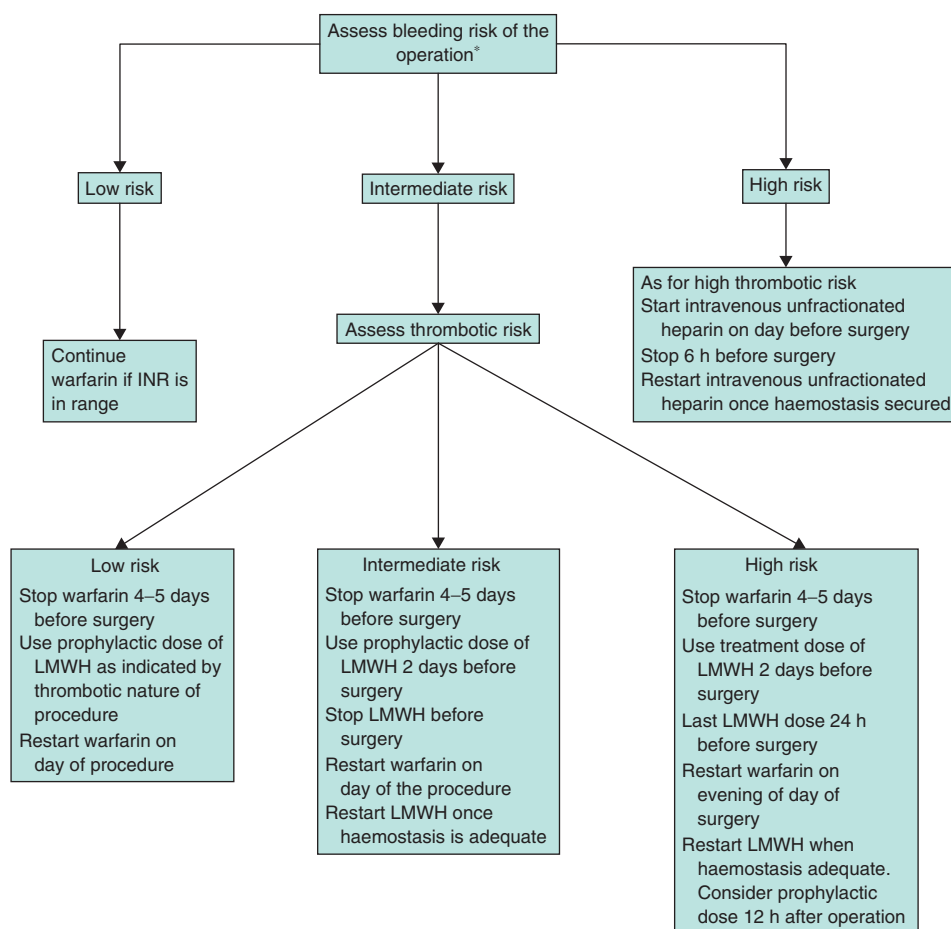
Fig. 1 Management based on risk assessment. *Bleeding risk is determined by the surgeon or operator (see *Table 2*). INR, international normalized ratio; LMWH, low molecular weight heparin

Stopping warfarin

Most surgical procedures can be performed safely if the INR is less than 1·5[23,24]. White and colleagues[25] found that if the INR is between 2·0 and 3·0 on warfarin, it almost always falls to less than 1·5 within 115 hours (4·8 days) from the last dose. So if the INR is between 2·0 and 3·0, four scheduled doses of warfarin could be withheld to allow it to fall spontaneously to 1·5 or less before surgery. It might be necessary to extend this period if the initial INR is high or maintained at a higher range (over 3·0) and also in elderly individuals. For operations with a high bleeding risk (such as neurosurgery), in which the INR should preferably be less than 1·2, a longer washout time may be required[25,26].

The decrease in INR over time is exponential and highly variable, and so INR testing is recommended on the day before surgery[25]. If it remains over 2·0, the administration of a low dose of oral vitamin K1 (1–2 mg) 24 h before the operation should be considered[27]. The

oral administration of vitamin K offers similar efficacy to intravenous administration, but with greater safety[27,28].

Starting heparin – 'bridging therapy'

'Bridging therapy' is the administration of an appropriate form of heparin while the oral anticoagulation is discontinued. In recent years, bridging has been procured mainly using LMWH, and only occasionally with intravenous UFH[14,18–20,29]. LMWH is started once the INR is less than 2·0 after the cessation of warfarin. For practical purposes, if warfarin has been stopped 4 or 5 days previously, LMWH injections can be started 36 h after the last warfarin tablet, without checking the INR, assuming that it has dropped below 2·0. This allows outpatient administration of the injections, avoiding unnecessary hospital stay. For patients with a high thrombotic risk, and for those who have erratic INRs, more frequent preoperative INR

Name:

Date of birth:

Address:

Anticoagulant drug …………………………...........................

Indication ……………………….................

Thrombosis risk

Bleeding risk

High ☐   Intermediate ☐   Low ☐   High ☐   Intermediate ☐   Low ☐

Reasons for categorization

  Thrombosis……………………………………………………................................

  Bleeding……………………………………………………………................................

Perioperative management

  Stop warfarin on date ………………………………….................

  Check INR on dates ....

  Start heparin on date …………………….....................

        Type ………………. Dose ………..........

  Stop heparin before surgery

        Date …………………….Time …………………............

  Restart heparin after surgery (check bleeding risk)

        Date …………………. Time ……………..........

  Restart warfarin after surgery

        Date ……………………. Time …………...........………

Copies of the form given to anaesthetist ◯ preop. ward ◯ postop. ward ◯ anticoagulation clinic ◯

Fig. 2 Perioperative anticoagulation chart. INR, international normalized ratio

checks may be necessary to assist in switching to LMWH if suboptimal anticoagulation is to be avoided.

LMWH is preferred over UFH because of its ease of administration and better pharmacokinetic profile.

Another advantage is that LMWH requires no monitoring in the absence of renal failure or pregnancy. In these two situations, monitoring may be performed with anti-Xa levels in discussion with a haematologist. Monitoring UFH

using the activated partial thromboplastin time (APTT) is often fraught with problems. Blood sampling is often not carried within the desired time period (6 h after start and/or change of the dose of heparin). Furthermore, the dosing of heparin according to the result is often poorly done, leading to under- or over-anticoagulation.

UFH might, however, be preferable in cases of excessive perceived bleeding risk (for example neurosurgical procedures, active or recent bleeding, and renal failure) mainly because of its short duration of action and its reversibility profile. UFH usually remains active for a maximum of 1 h after discontinuing the infusion if there is no associated renal impairment. It can also be reversed easily using protamine sulphate. A dose of 1 mg protamine sulphate neutralizes around 100 units of UFH; the dose of protamine is usually a maximum of 50 mg.

Stopping heparin before surgery

To avoid persistence of heparin during surgery, it is suggested that the last therapeutic dose of LMWH be given no less than 12 h before operation with a twice-daily regimen, or 24 h before operation with a once-daily regimen[18,30–33]. A recent study has shown that a 24-h gap for LMWH even when given twice daily may be preferable[34]. Intravenous UFH should be stopped 4–6 h before surgery[5].

Neuraxial anaesthesia

Several guidelines have been prepared on the management of neuraxial anaesthesia in patients receiving anticoagulants[35,36]. Most recommend that prophylactic treatment with LMWH should be stopped at least 12 h before the insertion of an epidural needle. Patients receiving treatment doses of LMWH require delays of at least 24 h to assure normal haemostasis at the time of needle insertion[36]. Removal of an epidural catheter should similarly be delayed for a minimum of 10–12 h after the last dose of LMWH. Subsequent LMWH dosing should occur a minimum of 2 h after catheter removal. If intravenous UFH is used, needle placement and catheter removal may be done 4 h after discontinuing heparin. Further heparin administration should be delayed for 1 h after needle placement[36].

Role of inferior vena cava filters

Prophylactic inferior vena cava filter placement may be considered if there is an extremely high risk of recurrent thromboembolic disease during the perioperative period. Examples include patients who have had an acute pulmonary embolism or a proximal deep vein thrombosis, and those with recent intracranial bleeding[37]. A temporary filter is generally preferred as it can be retrieved once the high-risk period is over. Discussion with both haematologist and interventional radiologist is recommended in these circumstances.

Postoperative period

Some degree of postprocedure bleeding may be acceptable, but it is imperative to remember that bleeding can also occur unexpectedly and without warning. Postoperative anticoagulation should be planned taking this bleeding risk into account. The initial management is with UFH or LMWH as warfarin takes a few days for full effect.

Restarting heparin and warfarin

If therapeutic-dose LMWH is being used, it should probably not be started until at least 24 h after surgery and only after haemostasis has been achieved[15]. A pragmatic approach would be to start LMWH at a prophylactic dose 12 h after operation and increase it over 36 h, especially in patients with high thrombotic risk. Twice-daily dosing may be preferable to once-daily dosing in the early postoperative period, as lower peaks of anticoagulant effect are achieved, and the smaller twice-daily dose is expected to be eliminated sooner if bleeding occurs close to the time of injection[19,31–33].

If intravenous UFH is chosen, it should be restarted without a loading dose at a rate of no more than 18 units per kg per h[38]. In the absence of a loading dose, the first APTT measurement should be deferred for 12 h in order for a stable anticoagulant response to be attained.

Warfarin may be restarted on the evening of surgery or whenever the patient is able to take oral drugs. The dose should be the same as the preoperative maintenance dose. Initial doubling of the maintenance dose for two consecutive doses after surgery is not evidence based and should be avoided. Once warfarin therapy is restarted, it can be expected to take at least 3 days for the INR to reach the usual therapeutic range of 2·0–3·0; this is due to its long half-life. If bridging anticoagulation is required, it should be continued until the INR is in the therapeutic range for 2 consecutive days[39]. It is also recommended that patients be monitored for bleeding once the anticoagulation has been resumed. Patients receiving either form of heparin should also have regular platelet counts to monitor for heparin-induced thrombocytopenia[40].

Emergency surgery

In the case of an emergency or life-threatening haemorrhage, it is important to reverse anticoagulation fully before operation. The product of choice is prothrombin complex concentrate (PCC). *Table 4* compares PCC with fresh frozen plasma (FFP). PCC can be administered rapidly without the need for matching the blood group or thawing the product, and has been shown in a number of studies to reverse warfarin-related coagulopathy[41–45]. The concentration of coagulation factors in FFP is less predictable, and many units do not contain sufficient levels of the four coagulation factors depleted by warfarin[46]. In contrast, the concentration of these vitamin K-dependent factors in PCC is approximately 25 times higher than in plasma, and so the volume of PCC required for reversal is significantly less[47]. The recommended dose of PCC is 25–50 units/kg[48]. A nomogram for PCC dose according to bodyweight of the patient, initial INR and desired INR is available[49]. Nearly 90 per cent of patients achieve the required INR within 15 min of PCC administration. A few INRs do not reach the desired level after a single dose and an additional smaller dose may be required.

The disadvantage of PCC is a possible increase in the incidence of thrombosis as it is a concentrate of vitamin K dependent coagulation factors. However, this phenomenon has been observed mainly in patients with haemophilia in acute surgical situations, and during warfarin reversal when other risk factors for thrombosis such as cardiomyopathy, shock and carcinoma were also present[44]. Recent studies have demonstrated that treatment with PCC in acute reversal of anticoagulation presents limited thromboembolic risk, and surrogate markers of thrombin generation did not increase[50,51].

If PCC is not readily available, FFP may be considered, although the problem of volume overload (recommended dose is 10–15 ml/kg) may be critical in patients with cardiovascular disease. In a cohort study by Makris and colleagues[42], in which urgent reversal of vitamin K antagonist was accomplished with PCC in 29 patients and with FFP in 12, complete reversal was not feasible with FFP when the INR was greater than 5 because of the large volume required. FFP may cause a lack of correction of factor IX, particularly in situations of over-anticoagulation (INR greater than 5), which can have implications in operations with high bleeding risk[42]. Recombinant activated factor VII (or VIIa) has also been shown to be safe, rapid and effective at reversing bleeding associated with warfarin anticoagulation. However, it does not seem fully to correct the warfarin-induced coagulopathy[52]. Further studies are required to establish a definite role for VIIa in these situations. It is imperative to administer intravenous vitamin K (5 mg) with all the products described above as these agents have a short half-life and the INR can climb again once their effect has worn off.

Management of perioperative antiplatelet therapy

The management of antiplatelet therapy in patients about to undergo surgery differs according to the clinical context. Aspirin irreversibly inhibits platelet cyclo-oxygenase 1, thus impairing platelet function. It may be given as primary prevention for cardiovascular and cerebrovascular diseases, in which case the drug can be discontinued 10 days before surgery to allow return of full platelet activity as the average lifespan of a platelet is 10 days. Recent evidence, however, weighs heavily towards the continuation of low-dose aspirin (less than 325 mg/day) unless there is a significant bleeding risk associated with the operation[53]. Clopidogrel binds irreversibly to the platelet receptor $P2Y_{12}$ thereby inhibiting platelet response to both exogenous and endogenous adenosine diphosphate. It is the second most commonly used antiplatelet agent, typically employed both as a substitute for aspirin or as an additional therapy for patients with unstable angina or after coronary stent implantation. If administered for primary prevention in place of aspirin, it can be discontinued 7 days before operation to allow active platelets to be present in the circulation[54].

Serious thrombotic risks are associated with the discontinuation of these agents when used as secondary

Table 4 Comparison between fresh frozen plasma and prothrombin complex concentrate

	Fresh frozen plasma	Prothrombin complex concentrate
Volume	Large	Comparatively less
Ease of administration	Hours	Minutes
Need for thawing	Yes	No
Coagulation factors	Variable (less effect on factor IX)	Concentrated factors II, VII, IX and X (those depleted by warfarin)
Risk	Volume overload, undercorrection, transfusion-related acute lung injury	Small risk of thromboembolism

prevention of vascular disease or after coronary revascularization. Recently, there have been recommendations from the European Society of Cardiology and American Heart Association regarding the duration of antiplatelet therapy for patients with coronary stent implantation (*Table 5*)[55,56]. Both these guidelines stress the importance of maintaining antiplatelet therapy in the immediate period after coronary stent insertion (at least for 1 year) to prevent stent blockage, which can have disastrous consequences. Burger and co-workers[57], in a meta-analysis of 2229 patients with drug-eluting stents, demonstrated that the premature discontinuation of antiplatelet therapy was the most significant independent predictor of stent thrombosis, with a mortality rate of 45 per cent. Patients who discontinue clopidogrel during the first month after coronary stent insertion are ten times more likely to die or to be readmitted to hospital during the next year[58]. The same applies to aspirin for a period as long as 15 months after percutaneous coronary stent insertion[59,60].

Although the above studies were conducted in a non-surgical setting, data from the postoperative situation have also demonstrated that the withdrawal of antiplatelet treatment can result in a very high mortality rate[61]. Schouten and colleagues[62] reported a series of 192 patients with drug-eluting stents, operated on within 2 years after stenting, either receiving aspirin and clopidogrel throughout the surgical procedure or with discontinuation of antithrombotic agents for 1 week[62]. They showed an association between early non-cardiac surgery after coronary artery stenting and perioperative adverse cardiovascular events specific to the discontinuation of antiplatelet therapy during the perioperative period. The risk of stent thrombosis associated with stopping antiplatelet agents is also influenced by factors such as the nature of the lesion and the timing of the procedure. It is likely to be highest when multiple recently implanted stents are present, particularly involving arterial bifurcations, and in patients with renal impairment, diabetes or dehydration[63].

The continuation of agents with an inhibitory effect on platelets may be expected to have associated bleeding risks. In the meta-analysis by Burger and co-workers[57], although aspirin increased the rate of bleeding complications by a factor of 1·5, it did not lead to a higher level of severity of bleeding complications, except in the situation of intracranial surgery and transurethral prostatectomy. The authors concluded that low-dose aspirin should not be discontinued before an intended operation or procedure unless there is a very high bleeding risk associated with it. However, this might not be reasonable for dual antiplatelet therapy with both aspirin and clopidogrel, which is known to increase the risk of surgical bleeding. A meta-analysis of five large randomized trials involving a combined total of over 75 000 patients has compared the bleeding risk of the combination of clopidogrel and aspirin with aspirin alone in patients at high risk of future cardiovascular events[64]. Adding clopidogrel to aspirin increases the relative risk of bleeding by as much as 50 per cent and the absolute risk by as much as 1 per cent. This risk remains increased in patients who stopped clopidogrel fewer than 5 days before surgery.

Until more trials are conducted, a risk-based approach may be considered. Patients requiring elective surgery and who are receiving dual antiplatelet therapy should, ideally, have surgery postponed until the recommended duration of clopidogrel therapy is finished (*Table 5*)[65]. If such a delay is unacceptable, the cardiologist, the surgeon and the anaesthetist should consider the balance of perioperative risk (for example stent thrombosis) compared with the possibility of increased surgical bleeding related to the procedure[66]. In situations of high bleeding and low thrombotic risk, the discontinuation of both clopidogrel and aspirin is logical. At the same time, in scenarios of high thrombotic and low bleeding risk, dual antiplatelet drug therapy may be continued until the day before surgery. If at all possible, continuation of at least aspirin should be considered[57,67]. In a case series of 15 patients with drug-eluting stents, Charbucinska and colleagues[68] suggested that continuation of aspirin and withdrawal of clopidogrel was not associated with severe postoperative complications. The operations included carotid endarterectomy and aortic aneurysm repair, and LMWH was administered in the absence of clopidogrel. However, a recent report claimed

Table 5 Guidelines on duration of antiplatelet therapy after coronary stent implantation

	Aspirin	Clopidogrel
Biomedical stent	Lifelong[55] At least 1 month[56]	At least 3–4 weeks[55] At least 1 month[56]
Drug-eluting stents	Lifelong[55,56]	3 months with sirolimus, 6 months with paclitaxel and at least 12 months if not at high risk of bleeding[55,56]

that aspirin therapy, in the absence of clopidogrel, does not completely protect the patient from the risk of adverse events and that the perioperative management of anticoagulation in such patients is difficult, needing more trials to guide appropriate management[69].

After operation, antiplatelet agents should be restarted as soon as is practicable and safe[70]. The precise timing should be discussed between the surgeon, anaesthetist and cardiologist. If there is a high risk of postoperative bleeding, restarting antiplatelet agents should be delayed until this risk has diminished, and removal of any indwelling catheters has occurred. Careful monitoring for cardiac ischaemia is imperative in patients who have drugs discontinued because of the high risk of coronary thrombosis.

In the emergency situation, platelet transfusion might be required to correct the thrombopathy induced by aspirin or clopidogrel because no pharmacological antagonists for these agents exist[66].

The American Society of Regional Anesthesia and Pain Medicine (ASRA) gives recommendations for the management of antiplatelet therapy in patients requiring neuraxial anaesthesia[36]. Aspirin does not represent an added risk for the development of spinal haematoma in patients receiving epidural or spinal anaesthesia. Complications have occurred after neuraxial blockade in patients receiving clopidogrel therapy, even though the medication was stopped 7 days previously[71,72]. However, these patients were also receiving LMWH which might have increased the bleeding risk. ASRA regards clopidogrel as an absolute contraindication to epidural anaesthesia. Regional anaesthesia in patients taking dual antiplatelet therapy also cannot be recommended and, in the emergency situation, a platelet transfusion should be given before the procedure[70].

Discussion

The perioperative management of patients receiving anticoagulation and antiplatelet agents can be problematic. It is important that the benefit of surgery is first weighed against the risk of altering the anticoagulation or antiplatelet regimen. Where doubt exists, there should be a discussion involving the physician managing the anticoagulation, the surgeon and the anaesthetist about the risks and benefits of continuing the anticoagulation or the antiplatelet agents. It may also be wise to involve the patient in the decision-making process and to consider an individual plan for complex situations. A multidisciplinary approach helps to manage the perioperative anticoagulation and antiplatelet therapy safely and effectively.

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